VIA MEDICA

LETTER TO THE EDITOR

Cardiology Journal
2014, Vol. 21, No. 4, p. 449
DOI: 10.5603/CJ.2014.0054
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ISSN 1897–5593

The double-edged sword of mechanical ventilation for patients with cardiogenic shock

Dr Lazzeri et al. [1] should be congratulated for their description of patients treated by percutaneous coronary reperfusion for ST elevation myocardial infarction (STEMI). They concluded that mechanical ventilation (MV) was an independent factor of short and long term mortality with quantitative relationship between MV duration and Intensive Care Unit mortality. However, quantitative relationship does not make causality. Furthermore, despite multivariate regression and propensity score analyses, their results are obscured by the most important missing variable, namely cardiogenic shock (CS) [2]. Reasons for intubation were ventricular fibrillation in 32 (30%) patients, pulmonary edema in 10 (10%) patients, and CS in 64 (60%) patients. In order to correct this bias, cardiac arrest and CS should have been introduced (or forced) into the model.

MV remained independently associated with mortality but not Killip class (a proxy for heart failure [HF]). However, these results deserve scrutiny as some patients may have been misclassified. The number of patients with Killip class III–IV receiving MV is 64, which is hardly possible with 64 patients with CS + 10 patients with acute pulmonary edema. From the 89 patients with Killip class III–IV who did not receive MV, only few must have suffered from CS as almost 7% of such STEMI patients present with CS in the literature and most of them must have received MV in their cohort: 64/1294 (5%) [2].

The precise role of MV in ventilator and hemodynamic assistance of patients with CS is sparse and not discussed in the paper. CS is characterized by low cardiac output along with decreased systemic vascular resistance: a stereotype of delivery/consumption (DO2/VO2) dependency. In order to improve this equation, means that increase DO2 or decrease VO2 are considered [3]. Unfortunately, most of treatments increasing DO2 have deleterious effects on outcome. Several means have the ability to decrease VO2 with various effects on outcome including:

1. Pain control but opiates have been associated with increased mortality in HF cohorts.
2. Lowering body temperature, which has been proved to limit infarct size and proposed for patients with CS.
3. Various degree of anxiolysis from light to profound sedation (± curarization) and MV that suppress respiration efforts accounting for 10–25% of global VO2.

However, MV has been consistently associated with nosocomial infection (including pneumonia in 23 of their patients), prolonged hospital stay and mortality. MV is hardly dissociable from treatments given for sedation and analgesia. Those agents (including propofol and remifentanyl used in their patients) have deleterious hemodynamic effects by decreasing already altered vascular resistance and immunologic effects, promoting infections. This could be counterbalanced by choosing a "cardio protective" analgo-sedative agent, for example volatile anesthetics. Moreover, positive pressure ventilation improves performances of dilated left ventricles with elevated filling pressure. To my knowledge, prognostic impact of MV in patients with CS is still not known. However, I would suggest reserving MV for [4]: (1) alteration of consciousness; (2) severe respiratory distress; (3) profound lactic acidosis; (4) rhythmic storm.

We don't need study to suggest that MV should be withheld in any patients, until not necessary. However, the risk/benefit of MV and its accompanying treatments is to be balanced in patients presenting with CS with no other definitive reason for receiving MV.

Conflict of interest: none declared

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Sébastien Champion, MD
Réanimation médicale et toxicologique hôpital Lariboisière, 2 rue A. Paré, 75010 Paris, France
e-mail: champion.seb@wanadoo.fr